THE ESTIMATION OF THE FUNCTIONAL POWER OF THE CARDIOVASCULAR APPARATUS.

A PRELIMINARY REPORT.

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Probably we are all fairly well satisfied with the ordinary methods for examining the heart and vessels in all cases in which, as we say, we *find something;* for example, in obvious valvular leaks, obstructions, and vascular degenerations. But there is a large group of cases in which we find nothing by auscultation and percussion, nor by the study of the palpable vessels and the examination of the peripheral parts and internal organs for evidence of passive congestion, even when these methods are supplemented by radioscopic examinations and blood-pressure measurements. "Heart negative" we often write; but we feel by no means sure that the heart is as sound as our examinations are negative.

Especially if we are interested, as no doubt we are, more in the question—"What can the heart do"—than in the question—"What anatomical lesions exist in it"—we need some supplement to the methods of examination above listed. In the differential diagnosis between cardiac neuroses and permanent organic damages of one or another description, between the nervous heart and the weakened heart, we are often in a quandary. Or, again, when we are not seriously in doubt about the diagnosis, but desire to know *how far* a patient's convalescence has advanced with respect to cardiac power, or *how far* a known lesion has weakened the cardiovascular apparatus, we sometimes find the ordinary methods of examination at fault. In such cases, following Rosenbach, we supplement them more or less by experiments. We say: "Work a bit, and see how much it tires you," or "Do you get out of breath when you go upstairs?" "How many pillows do you have to have at night?" "Do you have palpitation when you hurry?" These are very rough-and-ready methods, yet they sometimes yield us information of more value than our direct physical examination. In the attempt to render more accurate the data at which we aim by questions such as those just suggested, a number of procedures have recently been suggested by German writers. It is the purpose of this paper to describe some of these methods, and to report briefly our own experience with them.

I. The Effects of Changed Position. It has been noticed by Schapiro and others that the normal difference between the pulse rate in a standing position and the lower pulse rate in recumbent position disappears when the heart is seriously weakened. In 100 cases we have tested this simple procedure—by counting the pulse first while the patient stands, and then while he lies flat: we find that the normal slowing of seven to fifteen beats per minute, which recumbency produces in healthy persons, is diminished or altogether lost in cases of uncompensated valvular disease, and when the heart is seriously weakened by any cause. We have not yet studied this test extensively enough to know whether it would help us to recognize cases of *incipient* cardiac weakness not appreciable by the ordinary methods of examining the heart.

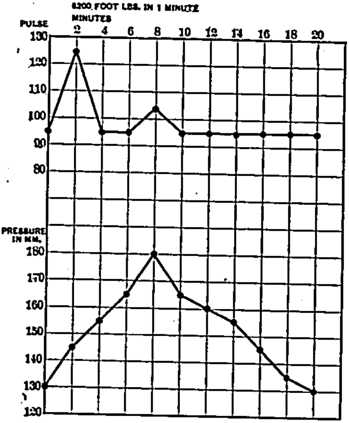

CHART I (which in our experiments represents the commonest type of normal reaction, or "*Erholung*" after exertion), shows a sudden rise in the pulse rate from 95 to 125 immediately following 6200 foot-pounds of work, accomplished in one minute by stair climbing. Four minutes after the performance of this work, when the pulse rate had fallen again to normal, the blood pressure had *risen* to 155, *or* 10 *mm. of mercury higher than it was when the pulse was most accelerated*. At the end of six minutes the pulse was still normal, but the blood pressure had risen to 165. At the end of eight minutes the blood pressure, continuing its rise, had reached 180 mm. After this a gradual fall in blood pressure occurred, the normal being reached only at the end of twenty minutes, although the pulse rate was normal in four minutes. Case 84,623. Normal. Aged twenty-four years.

II. HERZ'S TEST.—Another procedure, suggested by Herz,[1] is called by him the Self-checking Test (*Selbsthemmungsprobe*). He counts the pulse over a period long enough to assure a reasonably constant rate per minute. He then has a patient sit down and very slowly flex and extend the right forearm, putting all the while his full attention upon the movement, but endeavoring to avoid any contraction of the muscles. During this test the physician keeps his left hand supporting the patient's elbow, while his right hand, grasping the patient's wrist, directs the slow movements of flexion and extension, without, however, assisting or hindering them in any way.

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[1] Deut. med. Woch., 1905, xxxi, 215.

Herz asserts that while in normal hearts this procedure exerts no influence upon the rate of the heart, a weakened heart is notably slowed. We have tested this method on 125 cases of miscellaneous affections, such as are seen in our out-patient clinic, and in a general way we have been able to confirm Herz's results. We have found that the great majority of cases in which the cardiovascular system shows no abnormality when examined by the ordinary methods, are also negative to Herz's test. A certain proportion of hearts which by other methods show evidence of weakness, react as Herz describes to the self-checking test; that is, the pulse slows down from 5 to 20 beats. In carrying out the test,

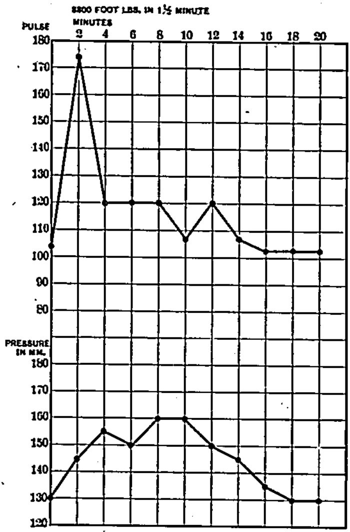

CHART II shows but slight variations from Chart I. In both of these cases the heart was young and apparently strong. Case 84,901. Normal. Aged nineteen years.

we have made it a rule that extension shall consume a full minute's time, and flexion also a minute. We are encouraged to follow this test further, although we have not as yet obtained any striking results with it.

III. LEVY'S TEST. Katzenstein's test[3] is based upon the well-known fact that if we increase the peripheral resistance opposed to the heart's action by compressing the abdominal aorta (in laboratory animals), a marked rise in blood pressure occurs. Anyone who is familiar with experiments on animals must have often seen the temporary resuscitation of a failing heart by compression of the abdominal aorta, and must also have noted that if the heart is quite exhausted, whether by hemorrhage or for other reasons, compression of the aorta is without its usual effect. In human beings

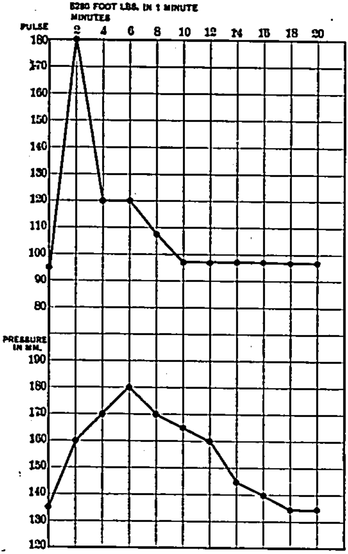

CHART III from a case of mitral stenosis, with perfect compensation, shows essentially normal conditions so far as this test is concerned. The rise in pulse rate, after 5280 foot-pounds of work performed in one minute, was more marked than in the normal heart, but the two charts are otherwise practically alike. Case 5354. Mitral stenosis. Aged twenty-one years.

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[3] Berl. klin. Woch., 1905, Nrs. 15 und 16.

Katzenstein imitates this familiar laboratory procedure by digitally compressing both femoral arteries at Poupart's ligament, and then noting the effects upon systolic blood pressure in the brachial artery. In normal persons this procedure is followed within two or three minutes by a rise of from 5 to 15 mm. of mercury in the systolic blood pressure. When the heart is hypertrophied (Levy[3] says) the rise is even more marked, while if the heart is weakened by any cause there may be little or no rise of blood pressure. Levy claims that no pain is produced by this procedure, but admits that it is useless in neurotic persons, since in them any such manipulation will suffice to raise blood pressure. We have made but few tests

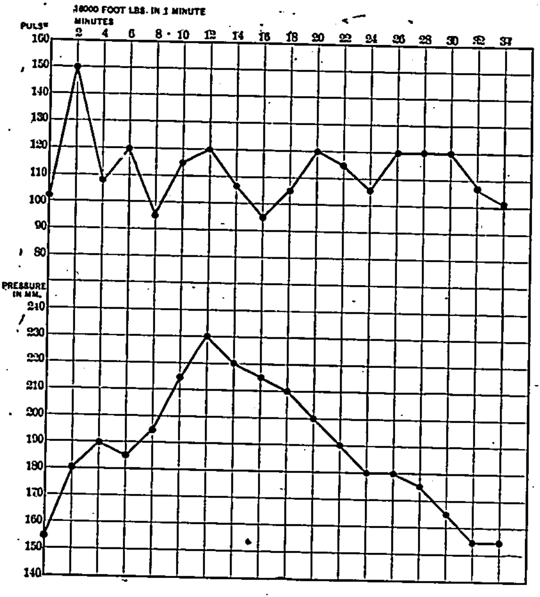

CHART IV shows the pulse and blood pressure curves from a case of weakened heart in a woman of forty-two. It will be noted that the "Erholung" is here *retarded*, so that the highest blood pressure is not reached until the end of ten minutes, while the return to normal occupies thirty-four minutes in all. Case 82,475. Myocarditis. Aged forty-two years.

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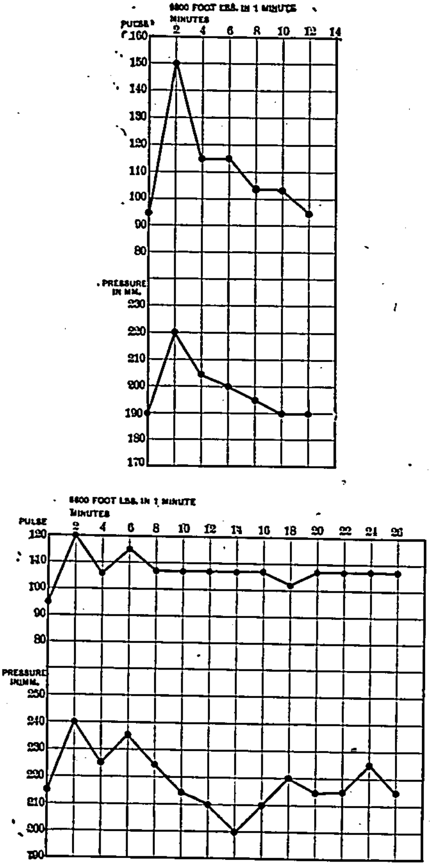
[3] Zeit. f. klin. Med., 1906, lx, 1 und 2.



CHARTS V and VI illustrate the curves which we have found in a number of more seriously weakened hearts in older persons, with higher blood pressure than any of those shown in the previous charts. In these two patients a slight rise in blood pressure was simultaneous with the rise in pulse; thence it gradually declined to normal in Chart IV, and slightly below normal in Chart V. The characteristic increase of blood pressure after the pulse rate has returned to the normal level is absent in these two charts. Chart V. Case 84,478. Arteriosclerosis. Aged fifty years. Chart VI. Case 84,516. Hypertension. Nephritis? Aged sixty years.

of this procedure, as we have been unable to perform it without causing considerable pain, the effect of which would naturally be to raise blood pressure independent of any other cause.

IV. Gräupner's Test. The test to which we have given the most attention is one of those elaborated by Gräupner[4] in several recent papers. Gräupner, who is a "Bad-Arzt" at Nauheim, found in the course of exercises prescribed as part of his treatment of cardiac cases that the reaction of weakened hearts after a measured amount of work differed distinctly from that of normal hearts. It is, of course, a familiar fact that the pulse rate and the systolic blood pressure rise after exertion, and that if exertion is sufficiently prolonged and arduous, both these figures may fall to or below the normal; but the phenomenon to which Gräupner has called special attention is quite different from this. Gräupner found that *after the pulse rate has risen and again fallen* to normal, as result of exertion, *the systolic blood pressure begins to rise,* reaches its maximum some minutes later than the pulse rate, and gradually falls thereafter to, or sometimes below, the normal. The phenomenon just described is that found by Gräupner in normal cases, and designated as the "*normal Erholung.*" In weakened hearts, if the damage is but slight, the "*Erholung*" still occurs, but is delayed and perhaps diminished in degree. In seriously weakened hearts there may be no "Erholung" whatever, the blood pressure declining from the start, then gradually re-ascending to the normal.

The main outlines of Gräupner's contention may be easily verified by anyone. Run quickly up two flights of stairs, and then stop and count your pulse. After the immediate acceleration is past or during the slowing of the pulse following it, you will note that the heart beat and the strength of the pulse become markedly exaggerated. One feels the "thump-thump" of the heart against the ribs much more strongly after the pulse has almost or quite reached its normal rate than during the period when the pulse is most accelerated.

In performing this test, Gräupner used a weight-and-pulley ergometer, but in our clinic this was not convenient, hence we substituted a measured amount of stair-climbing. We reckoned the number of foot-pounds of work by the height ascended, computed in feet and multiplied by the number of pounds which the individual weighed. The amount of work in foot-pounds has, therefore, varied considerably, according to the weight of the individual, although the number of stairs climbed has usually been the same. As regards the phenomenon designated by Gräupner as the "*normal Erholung,*" we can verify his findings, and we likewise agree with him in the results of our experiments upon seriously weakened hearts. In some of the cases believed by us from ordinary examina-

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[4] Collected in his monograph: Die Messung der Herzkraft, München, 1905, Otto Gmelin.

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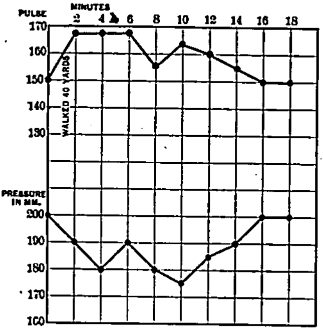

Chart VII shows the results obtained in a case of ruptured compensation, the patient being too ill to climb any stairs, markedly dropsical and cyanotic. Blood pressure *fell from the start* (simultaneously with a rise in pulse) and took eighteen minutes of perfect rest to bring it back to the (very high) point indicated at the beginning of the experiment. Myocardial weakness, cyanosis, etc. Aged forty-eight years.

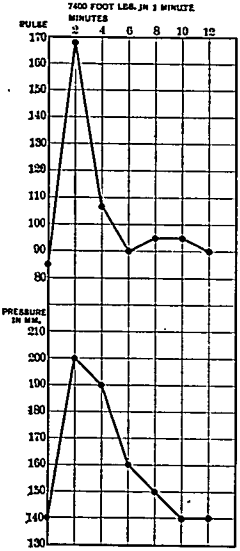

Chart VIII.—Case 84,318. Phthisis; "heart negative." Aged forty-five years, tion of the heart to be normal, there was considerable variation from the ordinary curve of blood pressure after exertion. On the other hand, various cases of valvular disease with good compensation showed, as might have been expected, a normal curve. (See Chart II.)

The important question regarding this and the other tests above described is, of course, "Do they help us to recognize the presence or the degree of cardiac weakening not appreciable by the methods already in general use?" We do not feel prepared to decide this question from the 75 experiments which we have been able to make in the four months during which we have been engaged in this work; but we have felt distinctly encouraged to continue the work, and in actual practice we find ourselves instinctively turning to Gräupner's test to verify our doubtful diagnoses of weakened heart, or to support us in the belief that the heart is normal when the patient has complained of subjective disturbances which might have been interpreted as evidence of passing congestion.

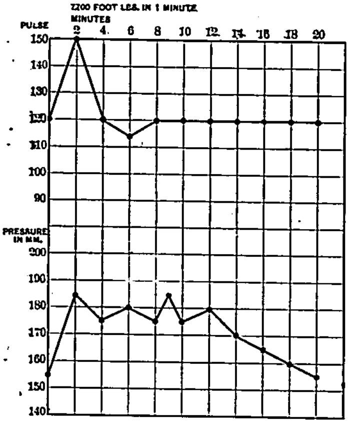

CHART IX.—Case 84,131. Palpitation. Nervousness. Myocarditis. Aged fifty-two years.

We suppose that the phenomenon just described depends primarily upon the amount of elasticity and contractile power in the arterial system; and, secondly, upon the ability of the heart to react upon the stimulus of increased peripheral resistance by beating more strongly. In all our measurements the effect of age in diminishing arterial elasticity is strongly suggested; but we have not yet seen reason to believe that this is the only factor concerned.

SUMMARY AND CONCLUSIONS.

1. For diagnosis, prognosis, and treatment it is important to supplement the current methods of cardiac examination by others planned to measure the functional capacity of the cardiovascular apparatus.
2. Several such methods have recently been suggested by Germans.
3. Our experience with these methods, and especially with that of Gräupner, leads us to hope that ground has been gained in the direction of functional cardiac diagnosis.

THE TREATMENT OF ILLUMINATING GAS POISONING BY THE DIRECT TRANSFUSION OF BLOOD.

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Occurrence. At least the main toxic factor in artificial illuminating (coal) gas poisoning is carbon monoxide. Carbon monoxide constitutes 6 to 10 per cent. of illuminating gas. It occurs in the so-called "water gas" to the extent of 30 to 40 per cent. It is the important toxic constituent of the "after damp" of mines, and of the gas from charcoal and coal stoves and from blast furnaces.

Mode of Action. Its action is purely that of asphyxia. It combines with hemoglobin, displacing the oxygen. Its affinity for hemoglobin is 300 times that of oxygen. The resulting compound is not perfectly stable, so that when oxygen is present in very great excess, carbonic oxidhemoglobin will be decomposed and oxyhemoglobin formed. Hence the rational treatment of patients by oxygen inhalation.

Haldane placed animals (mice) in oxygen under a pressure of two atmospheres, so that sufficient oxygen went into simple solution in the blood serum to support life independently of the hemoglobin. Sufficient carbon monoxide was present completely to saturate their hemoglobin. Under these conditions the mice remained normal as to symptoms, showing that the carbon monoxide has no direct toxic action. When the pressure was removed and the mice put out in the air they died with symptoms of asphyxia.

Haldane claims that in recovery carbon monoxide is given off from the lungs, as such, and is not destroyed or oxidized in the body.